AN EPIDEMIOLOGICAL STUDY ON CHRONIC BACTERIAL FOLLICULITIS

DISSERTATION

Submitted to
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
Chennai, in partial fulfillment of the university regulations for the award of

M.D. DEGREE IN
DERMATOLOGY, VENEREOLOGY AND LEPROSY
(BRANCH XII A)

DEPARTMENT OF DERMATOLOGY, LEPROSY AND INSTITUTE OF STD
THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI

FEBRUARY 2006

CERTIFICATE

We hereby certify that the work embodied in the dissertation "**AN EPIDEMIOLOGICAL STUDY ON CHRONIC BACTERIAL FOLLICULITIS**" is a record of work done by **Dr.SACHIN G. ROY**, in the Department of Dematology and Leprosy and Institute of STD, Madras Medical College, Chennai, during his postgraduate course from 2003 – 2006. This work has not previously formed the basis for the award of any degree or diploma.

Dr.Kalavathy Ponniraivan, B.Sc., M.D.,
The Dean
Madras Medical College,
Chennai – 600 003.

Prof.Dr.N.Gomathy,M.D., D.D.,
Professor and Head
Department of Dermatology & Leprosy
Madras Medical College,
Chennai – 600 003.

SPECIAL ACKNOWLEDGMENT

I greatfully acknowledge and sincerely thank **Dr. Kalavathy Ponniraivan, B.Sc., M.D.,** *Dean, Madras Medical College and Government General Hospital, Chennai – 600 003, for granting me permission to utilize the resources of this institution for my study.*

ACKNOWLEDGEMENT

I am gratefully indebted to **Dr.N.Gomathy, M.D., D.D.,** Professor and Head of the Department of Dermatology for her invaluable guidance motivation and help through out the study I would like to express my sincere and heartfelt gratitude to **Dr.B.Parveen, M.D.,D.D.,** Additional Professor, Department of Dermatology for her constant support and motivation. I sincerely thank **Dr.C.Janaki,M.D.,D.D.,** Reader, Department of Dermatology.

I express my earnest gratitude to **Dr.V.Somasundaram, M.D.,D.D.,** Additional Professor, Department of Occupational Dermatology and contact dermatitis for his help and support.

I am very grateful to **Dr.N.Usman, M.D.,D.V., Ph.D.,** Director, Institute of STD for his immense cooperation and help. I also thank **Dr.V.S.Dorairaj, M.D.,D.D.,** Additional Professor, Institute of STD for his kind help. I wish to thank **Dr.S.Mohan, M.D., D.V.,** Registrar, Institute of STD for his support.

I express my sincere gratitude to **Dr.K.Rathinavelu,M.D.,D.D.,** Professor of Leporsy and **Dr.R.Arunadevi,M.D.,D.D.,** Lecturer / Registrar, Department of Dermatology for their support.

My sincere and heart felt thanks go to **Dr.K.Manoharan, M.D., D.D., Dr.R.Priyavathani, M.D., D.D., D.N.B., Dr.V.Anandan M.D., (Dermatology), D.C.H., D.N.B (Paediatrics) Dr.V.Sampath, M.D., D.D.,** and **Dr.G.K.Tharini, M.D.,** Assistant Professor Department of Dermatology of immense help, kind support, constant motivation and encouragement they showered on me.

I am grateful to **Dr. A. Hameedullah, M.D.,D.D., Dr.Thirunavukkarasu, M.D., D.D., Dr. R. Suganthi, M.D., Dr. J. Manjula, M.D., D.N.B., (Dermatology),** Assistant Professors for their encouragement.

I wish to thank **Dr.S.Venkateshwaran, M.D., D.V., Dr. Ilangovan, M.D., D.V., Dr. Thilagavathy, M.D., Dr. Thirunavukkarasu, M.D., D.V., Dr.Ramachandra Reddy, M.D., D.V., Dr.P.Mohan, M.D.,D.V.,** and **Dr.S.Arunkumar, M.D.,D.V.,** Assistant Professor, Institute of STD, for their help and suggestions.

I am also thankful to **Dr.V.R.Janaki, M.D., D.D., MNAMS, Dr.G.Sentamil Selvi, M.D.,D.D.,MNAMS, Ph.D.,** and **Dr.Jeyakumari Jeevan, M.D., D.D.,** for their continuing guidance and support.

I duly acknowledge the paramedical staff and my colleagues for their help and favours.

Last but not least I am profoundly grateful to all patients for their cooperation and participation in the study.

CONTENTS

INTRODUCTION

Since ancient times pyogenic infection is one of the commonest entity affecting man. It is especially more common in developing and the underdeveloped countries.

Factors like overcrowding, malnutrition, improper hygiene predispose to pyodermas. It has chronic course due to difficulty in achieving a long lasting cure.

Herewith two entities viz dermatitis cruris pustulosa et atrophicans, and Sycosis Barbae are dealt. An attempt is made to cater to the epidemiology of the said entities, which are some of the common causes of chronic morbidity due to bacterial infection.

REVIEW OF LITERATURE

Fundamentals of cutaneous microbiology[1,3]

The normal human skin is colonized by huge numbers of bacteria that live harmlessly as commensal on its surface and within its follicle. Infection develops when the right combination of causative factors exist.

Normal flora of the skin[1]

1. Residents
2. Temporary Residents
3. Transients

Species belonging to the normal resident flora category [4]

These include

1. Micrococcaceae (non coagulase staphylococci, micrococci)
2. Aerobic diptheroids (Corynebacterium, Brevibacterium)
3. Anerobic diptheroids (Propionibacterium)
4. Lipophillic yeast pitryosporum
5. Enterobacteriaceae
6. Other bacterias

Kloos and Schleifer scheme is now used to classify micrococcaceae rather the previously used Baird – Parker System[2]

Within the genus, Staphylococcus, ten different species have been regularly isolated from normal skin. S.epidermis is the principal staphylococcal spp, among coagulase negative staphylococci, Staph epidermis – (Principal Staphylococcal spp)[5], S hominis – (2nd MC)[5], S capitis, S cohnii, S haemolyticum, S saprophyticus, S warneri, S xylosus have been isolated.

Staphylococcus aureus is not considered as resident on healthy skin although in one third of population it isolated from anterior nares[1].

FLORA OF SOME SPECIALISED AREAS [1]

Certain areas of skin have specific flora which differs qualitatively and quantitatively, which deserves mention.

a. NASAL VESTIBULE

The commonly found organism by swabbing of anterior nares is coagulase negative staphylocci, coryneforms, S aureus is present as resident or rather contaminant in about 35% of healthy subject[2].

FACTORS INFLUENCING THE DISTRIBUTION AND LOCALIZATION OF BACTERIAL FLORA[1].

Bacterial flora of the skin varies according to age[6,21] sex and racial factors. There is evidence that males carry higher number of aerobic bacterias than female.[2] Racial differences have been found in nasal carriage of S aureus in children [7]. In white individuals (at 41% positivity), black individuals (30%positivity).

Other factors

Washing – Abstinence from washing does not increase the count.

A scrub bath temporarily redistribute the colonies leading to apparent reduction in density of colonization[2]

Season – External temperature and humidity in combination has shown to affect localization[8].

Antiseptics – Antiseptics applied to skin generally removes transient flora and temporarily reduce resident organism [9]

BACTERIOLOGY AND PATHOGENESIS

(Epidemiology)[10]

Skin offers a good medium for growth of organism by virtue of protein, fats, carbohydrate derived as byproducts of keratinisation and water and minerals from sweat and sebaceous glands.

The ubiquitous presence of S.aureus and the difficulty in distinguishing among strains has made understanding the epidemiology of staphylococcal infection difficult.

Epidemiology studies utilizing phage typing techniques suggest that transfer of organism occurs via hands of personnel. Individual with open staphylococcal infection are dangerous potential carriers and transmitter of infection. Colonization of S.aureus may be transient or represent a prolonged carrier state S.aureus produces many cellular components and extracellular products that contribute to pathogenicity. Coagulase, leucocidin and exotoxin have been implicated in pathogenesis. Host factors such as immunosupression, glucocorticoid therapy, atopy and preexisting tissue injury or inflammation are a major determinants in pathogenesis of staphylococcal disease. S.aureus pyoderma occurs in individual who are nasal carries of the organism, which when translocated onto the skin, is able to gain access via small breaks in the cutaneous integrity and cause superficial infection.

The high prevalence of staphylococcal infection is substantiated by universal presence in adults of circulating antibody to one or more cell wall antigen or extracellular toxins. The occurrence of infection despite circulating antibody suggests that hypersensitivity may play a role in recurrent staphylococcal

infection. Adhesin such as lepotechoic acid promotes close opposition between staphylococci and human cell.

Skin and defence[(1)]

Skin has emerged as a remarkably talented immunological organ. Kligman et al- stated that primary factor determining the number and kinds of organism found on skin depends on the availability of water[(1)].

Defence depends on:-

1. The nature and health of the epithelial surface including its ability to replicate and produce secretions
2. The interaction between commensal organism of normal flora and the potential parasite.
3. The cellular and humoral factors acting within the body – both classical immune mechanism and nonspecific mechanism.

Several mechanisms operate in skin to eliminate pathogenic organisms.

Periodic desquamation, desiccation, drying prevents colonization. The negative electrical charge of skin is quite effective again S aureus colonization.

Ricketts et al stated that chemical mechanism and drying was responsible for elimination of S aureus[11]

Lacey et al confirmed that extraction of fatty acid from skin diminishes resistance[12].

Shinefield et al proved the role of bacterial interference in colonization of skin [13].

Selwyn et al showed the importance of antibiotic production by coagulates – negative staphylococci and lipophilic diptheroids[14]

Micro organism interact and activate the serum complements to generate chemoattractants that recruits phagocytes and opsonin [3]. They can bring about the activation of antigen – antibody complex. They can induce arachidonic acid metabolism by damaging host membrane and can initiate influx of inflammatory cells through lipoxygenase dependent pathway (leukotriene Bu)

STAPHYLOCOCCUS AUREUS(15)

Staphylococcus aureus is an aggressive pathogen and most common cause of primary pyodermas, soft tissue infection and secondary infection.

Morphology[16] - Staphylococcus aureus is a gram positive organism. Spherical cells (1µm) usually arranged in grape like pairs, tetrads or chains is seen. Young cocci are strongly gram positive and older cultures become gram negative.

Cultural characteristic[16] – S.aureus grow readily on most bacteriological media under aerobic and micro-aerophillic condition. Colonies on solid media are rounded, smooth, raised and glistening. S.aureus usually form grey to golden yellow colonies. Various degrees of hemolysis is produced by S.aureus.

Biochemical characteristic[16] – S.aureus is coagulase positive which differentiates it from other species. Coaglase plays role in the forming of Staphylococcus abscess.

Specific sites carriage rate[5]

Anterior nares- 35%
- 20% - Persistent nasal carriers
- 60% - intermittent
- 20% - resistant to nasal colonization

Nasal carriage state[5]

Genetic factors involving bacterial adherence and immune response are implicated in nasal carriage rate[9]

In nasal passage anterior nares and mucosa of turbinates are readily colonized

Higher nasal carriage rates are seen in

1. Atopic eczema[17,18]
2. Phenylketonuria[19]
3. HLA – DR_3[20]

Carriage of S aureus is physiological and itself not an indication for treatment.

Permanent eradication of carriage of S aureus is not possible.

HOST DEFENCE FOR S. AUREUS[22]

Main host defence against staphylococci is ingestion and killing by phagocytes.

Cell medicated response to S aureus occur but role is uncertain antibodies are of limited diagnostic and prognostic value[23].

Conditions with increased risk of S aureus infections are :

1. Poorly controlled diabetes mellitus[24]
2. Immunosuppression therapy
3. Renal insufficiency
4. Job syndrome
5. Buckleys Syndrome[25]

6. Wiskott Aldrich Syndrome[18]
7. AIDS

FOLLICULITIS [26]

It is an infection of hair follicles. It can be classified according to

1. Level of involvement of hair follicle
2. According to microbiological etiology

1. Level of involvement of hair follicle[26]
    a. Superficial folliculitis Infection at the level of follicular ostia)
        i. Bockharts impetigo (follicular impetigo)
        ii. Chronic folliculitis of leg
        iii. S.aureus blepharitis
        iv. Pseudomonas folliculitis
        v. Gram Negative folliculitis
        vi. Pitryosporum folliculitis
        vii. Acne necrotica miliaris
        viii. Pseudofolliculitis (pili incarnati)
        ix. Physical
            a. Traumatic folliculitis of epilation
            b. Folliculitis beneath adhesive tape
        x. Occupation – mineral oil or tar induced folliculitis

xi. Actinic folliculitis

xii. Disseminate and recurrent infundibulofolliculitis

xiii. Trunk folliculitis

xiv. HIV Associated

a. Eosinophilic pustular folliculitis

b. Itchy folliculitis

xv. Folliculitis of pregnancy

xvi. Steriod induced folliculitis

b. Deep Folliculitis (Infection at the level of hair bulb)[26]

i. Sycosis Barbae, Lupoid sycosis

ii. Furuncle

iii. Carbuncle

iv. Pyoderma faciale

v. Folliculitis declavans

vi. Kerion

vii. Favus

viii. Majocchis Granuloma

ix. Acne Keloidalis

x. Acne conglobata

xi. Perforating folliculitis

xii. Dissecting cellulites of scalp

xiii. Hidradenitis Suppurativa

2. According to Microbiological Etiology

Bacterial Folliculitis[27]

i. S.aureus folliculitis – Bockharts impetigo
Chronic folliculitis of leg
Sycosis Barbae
Furuncle
Carbuncle

ii. Pseudomonas - Hot tub folliculitis
Swimmers ear

iii. Gram negative folliculitis (Klebsiella, Enterobacter, Proteus) - Commonly seen after long term treatment of acne with antibiotic.

iv. Syphilitic folliculitis

Viral Folliculitis - Herpes simplex folliculitis
Follicular molluscum contagiosium
HIV associated folliculitis

Fungal -

Dermatophyte - Tinea capitis
Tinea barbae

Majocchis granuloma

Candidal folliculitis

Pitryosporum folliculitis

Infestations

Demodicidosis

Helminths

Schistosoma - Swimmers folliculitis

Hook worm folliculitis

SYCOSIS BARBAE [28]

Synonyms

Non Scarring Sycosis Barbae [29]

Shave bumps

Razor bumps

Barbers itch

Barbers rash

Folliculitis barbae traumatica

Chronic Sycosis Barbae

Sycosis vulgaris

Aliberts disease III (Aliberts mentagra) [30]

Scarring Sycosis Barbae[28]

Lupoid Sycosis

Ulerythema Sycosiforme

History:

The word sycosis is derived from Latin element meaning ‘small fig’[31]. First description of Sycosis Barbae was made by J.L.Alibert under the name Aliberts mentagra in 1825[31].

Definition :

Sycosis is a subacute chronic pyogenic infection involving the whole depth of follicle involving the beard areas of face and upper lip [28,32]

Epidiemiology [28]

Age & Sex

Sycosis occurs only in males after puberty and commonly involves the follicles of the beard.

Most cases begin in 3rd and 4th decade. There is a report of sycosis in a boy aged 14 years.

Prevalence

It was commoner when more men wore mustaches or bear in preantibiotic era.

Aetiology

The infecting organism is staphylococcus aureus the same phage type which can be isolated from nose [33].

Staphylococci generally do not penetrate beyond the follicular ostia. So some unknown constitutional factors may determine the susceptibility of staphylococci to penetrate beyond follicular ostia.

Predisposing Factors

1. Many patients are seborrheic with greasy complexion and chronic blepharitis[28]
2. Emotional stress (may be precipitating)
3. Trauma due to shaving
4. Sharing towels and face washers
5. It is seen that clinical and other indoor workers are affected more than those who work in open air[28]

Pathology

The affected follicle is packed with polymorphonuclear leucocytes, which infiltrates its wall. Around the follicle there is chronic granulomatous infiltrate in which lymphocytes, plasma cell, histocytes and foreign body giant cell are conspicuous. The sebaceous gland, or the whole follicle, may be destroyed and replaced by scar tissue especially in lupoid sycosis[28].

In some instance, epithelium grows down from the surface to encase both the hair and the inflammatory response, assisting their eventual transepithelial elimination[34].

In healed cases atrophy of epidermis and hair follicles and fibrosis of dermis is present.

CLINICAL FEATURES

Seen in male having moustaches and beard mostly in middle age.

Sites involved [35]

Upper lip (Moustache area)
Angle of mouth
Whole beard area

Symptoms

1. Itching is the prominent symptom
2. Burning sensation
3. Patient gets uncomfortable sensation and finds it very difficult to shave.

4. Patient also complains of cheesy white maternal discharge from the lesion

Sycosis Barbae passes through 3 stages

1. Acute stage
2. Subactue stage,
3. Chronic stage
    a. Without scarring
    b. With scarring (lupoid sycosis)

Acute stage [35]

The essential lesion is an oedmatous, red, follicular papule or pustule centered on a hair. Initially the individuals papules remain discrete neglect leads to spreading to adjacent follicles leading to coalescing of the perifollicular oedema. It produces raised plaques studded with pustules classically resembling its appearance to ripe fig for which it was coined as ‘sycosis’.

Subacute Stage[35]

Subacute stage is more commonly seen. The lesion may be scattered irregularly over the beard, or grouped especially on the upper lip and below angles of the jaw.

Chronic Stage[35]

(a) Without scarring

Attacks of varying duration occurs at irregular intervals over months and years. In more chronic forms the lesions are typically clustered into plaques and may persist for very long period. A case of nearly 20 years chronicity has been reported [36].

There is often some crusting and scaling and impetiginisation. Vegetative, almost granulomatous reactions are not unknown especially in the moustache area. The hair neither fall nor is the growth retarded despite the inflammation.

b. With Scarring

Sometimes Sycosis Barbae enters its chronic scarring form for which the term ‘Lupoid Sycosis’ is given

It is a variant of Sycosis Barbae that destroys the hair follicles and produces extensive scarring. Its distinguishing characteristic is a perisisting, slowly enlarging circinate patch occurring any where on the bearded area, on the temples or under the chin.

Clinically it is seen as a pink atrophic scar with permanent hair loss, surrounded by papules and pustules over the advancing margin. The atrophy is consequence of a prolonged deeply dissecting folliculitis, which destroys the hair bulb and devitalizes the deeper corium. Pitted scars may be seen in the central atrophic zone. The disease eventually burns itself out after some years.

Lupoid Sycosis, despites it name resemble lupus vulgaris or lupus erythematous only remotely lupoid sycosis beginning high on face may extend into the scalp. This behaviour suggests a relationship with folliculitis declavans.

Rarely a similar process affects axillary pubic hair, lower legs, thigh or arms.

DIFFERENTIAL DIAGNOSIS

1. The most frequent misdiagnosis is certainly pseudofolliculitis barbae[29]

In many literature pseudofolliculitis barbae and Sycosis Barbae are considered as one and the same entity.

However following difference are worth mentioning

A. Pseudofolliculitis barbae principally occur in men who shave

B. It is generally seen in darkly pigmented skin (of African descent) having tightly curled hair.

2. Tinea barbae (Mycotic Syscosis), kerion of beard, usually occurs on chins and cheeks. T rubrun and T mentragrophytes are commonly implicated. It can be only confirmed after microscopic examination (Koh) of skin and hair scrapings [37]

3. Herpetic sycosis

4. Acne vulgaris can some times be mistaken

5. Klebsiella oxitoca folliculitis resembling Sycosis Barbae (tinea barbae) has been reported in a 55 year old male, dairy farmer who was a known diabetic[59].

DERMATITIS CRURIS PUSTULOSA ET ATROPHICANS (DCPA)

Synonyms

1. Clarke's disease[38]
2. Folliculitis depilates despartes glabre[39]
3. Epilating folliculitis of glabarous skin[40]
4. Folliculitis cruris pustulosa et atrophicans [41]
5. Nigerian skin disease[42]
6. Segmental folliculitis [43]
7. Chronic folliculitis of leg[44]
8. Dermoepidermis microbeinns[45]
9. Pustular dermatitis atrophicans of leg[38]

History

Follicular disease of glabarous skin is known since ancient times.

First authentic reports were in French literature by Dubreuilh W. in 1895 and Arnozan X in 1897 under the term Folliculitis depilantes desparates glabres[39].

Clarke in 1952 gave the term in Dermatitis Cruris Psutulosa et atrophicans among negroes in Nigeria[38].

Harman used the term Nigerian Skin disease in 1968[42]

In India in 1964 Desai SL et al described it as a pyogenic folliculitis of leg with associated hypergammaglobulinaemia[46].

Sugathan P et al described in 1973 described a similar condition[41].

Definition : It is chronic pyogenic infection of hair follicle of the glabrous skin, involving most commonly the lower limb. It starts as a superficial follicle later on involving the whole depth of follicle.

Epidemiology

Age & Sex :

Dermatitis cruris pustulosa et atrophicans was described by Clarke as occurring in adults. Male predominance was recorded by Clarke[38]. In female patients well developed terminal hair over the shin was the prerequisite.

Seasonal distribution

Peak incidence in summer month is seen.

Geographical distribution

It was first reported from black African countries of Nigeria in Laos by Clarke.

Aeitiology[38]

Bacterial infection with staphylococcus aureus as main causative agent has been proved regularly by culture studies staphylococcus albus has also been reported.

Predisposing Factors

External application of

a. Strong soaps and detergents [47]
b. Coconut oil and palm oil, petroleum jelly

Trauma by : Pumice stone[39], walking through long grass[42], coarse stockings, contact with cow dung

c. Ingestion of animal proteins, alcoholic drinks[41]
d. Excessive sweating
e. Aggravation in summer
f. Psychological stress and sleeplessness aggravates the condition[41].

Pardo Castello described association of folliculitis in sugarcane cutters suggesting an injury with sugarcane needles as possible precipitating cause[48].

Coarse stockings or wearing long trousers especially in Army troops has shown as a predisposing cause.

Histopathology

Lever describes subcorneal pustule in hair follicle, perifollicular infiltrate comprising predominantly of neutrophils is seen involving upper portion of hair follicle. Inflammatory infiltrate is seen perivascularly and around eccrine ducts.

Some cases acanthosis and parakeratosis were seen[49] Clarke description of moderate acanthosis, parakeratosis with polymorphous infiltration is recorded. He described papillary oedema, subepidermal infiltration with lymphocytes and plasma cells[38].

According to Harman histopathological features can be described as active and healed case. Healed cases mostly showing atrophy of epidermis and hair follicle with fibrosis of dermis[42].

Sugathan et al described in three stages namely early, well developed and cicatricial stages[41].

Miller described similar features with foreign body giant cells perifollicularly, absence of elastic fibres within the cellular infiltrate and collagen fibres well atrophic and sclerosed in late stage[40].

Clinical Features[41]

It is a disease commonly occurring in adults with male predominance. Women who show well developed terminal hair over shins are more prone. Dermatitis cruris pustulosa et atrophicans is a disease affecting the glabarous skin.

Sites involves

1. Lower legs mostly over the shins (most common site), it may involve bilaterally (commonly) as well as unilaterally (rarely).
2. Upper thigh
3. Dorsum of forearms
4. Upper arms
5. Dorsum of foot, scalp, trunk, and pubic hair are almost never involved.

It has some typical features.

1. Peculiar affinity to legs
2. More or less symmetrical involvement.
3. Atrophy of the skin
4. Inevitable alopecia
5. Extreme chronicity (flare ups and remission)

Symptoms

Most common and important symptom is itching. Others may complain of burning sensation, pain. Symptoms vary in severity but most of the patients have an uncomfortable sensation. The patients generally do not have any systemic complaints.

General health of patient is good.

Clinical grading has been given by Sugathan et al[41]

Grade 1 : Follicular pustules with few of them having a well defined perifollicular erythema

Grade 2 : Predominant lesions are follicular pustules with significant number of infiltrated papules around broken hair. The papules show excoriation marks, crusting a

peripheral rim of white scales. Alopecia is present but not marked. Wiry roughness is felt due to broken or irregular hair.

Grade 3: Characterized by almost complete alopecia with atrophy shiny and scaly skin. Pustules are not seen but few discrete scales are seen at periphery.

The disease passes into 3 stage[41]

1. Early active stage
2. Subacute stage
3. Cicatricial or burnt out phase

1. Early active Stages[41]

The onset is commonly seen to start from lower third of legs almost simultaneously[42]. Sometimes it may start unilaterally followed by bilateral involvement.

Borders especially lower one are well defined conforming the lower limit of terminal hair at ankle. It spreads upwards to almost always involve the anterior tibial surface of leg. Dorsum of leg is never involved.

Nearly all follicles in affected area contain a pustule centered over the follicle. Perifollicular erythema can be appreciated.

2. Subacute stage[41]

Untreated active stage passes into this stage. Follicular pustules along with numerous infiltrated papules. Excoriated mark are seen with crusting and scaling over the lesion. Cutaneous oedema is evident by inability to pinch the skin. Wiry roughness with reduction in hair bearing follicle.

3. Cicatricial stage[41]

It is characteristed by complete alopecia with paper atrophy of the skin while the disease has completely burnt out in one area active lesion at other areas is not uncommon.

In general no hypopigmentation or hyperpigmentation is seen.

TREATMENT FOR DCPA AND SYCOSIS BARBAE [15,28]

Both of these conditions are medically managed and more or less have similar treatment modalities.

Pus Culture and Sensitivity – It is advised in all cases with pustular lesions.

Nasal Swab – Is done to detect carrier state but not routinely advised. Underlying malnutrition, anemia, nutritional deficiency,

diabetes, immunodeficiency if associated should be treated simultaneously.

Treatment divided into

a. Treatment of carrier state

b. Treatment of disease proper

A. Treatment of carrier state

If nasal swab indicates a carrier state then topical or oral antibiotic can be used.

a. Topical – Intranasal 2% mupirocin for 5 days in all patient. 50% people remain organism free for 5 months [50].

Other topical antibiotic like neosporin can be used.

b. Bacterial interference – Nasal inoculation of S aureus strain 502A can be used artificially for achieving bacterial interference against more virulent S aureus strains [15].

c. Staphylococcal vaccine – Derived from lysed bacteriophage exotoxin to boost up humoral response.

Oral – Rifampicin 600mg/day for 7 days cleared organism for 3 months in 80% cases [51]. Clindamycin at dose 150mg/day can be used [52].

PROPHYLACTIC MEASURES

Precaution

1. Patients with DCPA should be asked to avoid abrasive pumice stone, external application of oil.
2. In person with Sycosis Barbae, shaving should be avoided. Hair can be clipped short.

Hygiene

Local hygiene in treatment of affected area is of outmost importance. Gentle daily cleaning with germicidal soap (chlorhexidine gluconate) is advisable. Application of warm compresses with saline or burrow solutions is effective. 6.25% aluminium chloride hexahydrate which has an antibacterial effect is useful [15].

TREATMENT OF DISEASE PROPER

Topical Therapy

2% mupirocin, neosporin, nadifloxacin, clindamycin fusidic acid, bacitracin has all been used topically with some success.

3 – 10% silver sulfadiazine cream has also found to be effective. Opening of pustules with sterile needle with application of boric acid [53] or 1% gentian violet paint has been found to be effective.

Oral Therapy[54]

Oral antibiotics depends upon the pus culture and sensitivity.

Cephalasporins	(1g/day PO for 10 days)
Cloxacillin	(1-1.5g/day PO for 10 days)
Doxycyline	(100 mg/day PO for 10 days)
Erythromycin	(1 g/day PO for 10 days)
Sulfadiazine	(4 g/day PO for 12 – 15 days)
Clindamycin	(150 – 300mg / day)

Sequential Therapy

Cotrimoxazole Ds twice daily with rifampicin 600 mg/day for 10 days have been found to be effective [55].

Puvasol

Using 8 methoxypsoralen orally and sun exposure for 15 minutes with cotrimoxazole Ds twice day for 10 days has been found to be effective [55].

This treatment has been especially useful in lupoid sycosis.

Systemic steroids with antibiotic can be tried in acute exacerbation.

AIMS OF THE STUDY

Aims of the study were purely epidemiological

1. To study age and sex distribution of patient
2. To study duration and seasonal variation chronic bacterial folliculitis
3. To know precipitating factors
4. To assess microbiological aspect of the disease
5. To determine the sensitivity pattern of microorganism to various class of antibiotics

MATERIALS AND METHODS

SELECTION OF CASES

a. 50 cases of Dermatitis cruris pustulosa et atrophicans, 25 cases of Sycosis Barbae were selected over period of 16 months from January 2004 to April 2005 for study attending the Department of Dermatology and STD at Government General Hospital, Chennai.
b. There was no exclusion criteria
c. Diagnosis of all these cases was mainly clinical.
d. Subsequently careful history was elicited with particular reference to the following.
    i. Age, Sex, occupation and socioeconomic status
    ii. Site, morphology and distribution
    iii. Seasonal variation, duration and mode of onset and progress
    iv. Symptoms related to each disease entity
    v. History external oil application

All cases were thoroughly examined for specific skin lesions and for associated skin or systemic disorder.

Following investigations were done in all cases

a) Complete haemogram
b) Pus culture and sensitivity in all cases (pustular lesions)
c) Gram staining for all pustular lesion
d) Blood sugar

Additional Investigations

Scraping for fungus and tzanck smear for herpetic sycosis was done where different diagnosis was suspected.

Biopsy was done only for selected cases where diagnosis was in doubt for exclusion.

OBSERVATIONS

Age Distribution

Age distribution in the study varied from 16 years to 52 years. (75 cases were registered).

Table I – Gives the Distribution 50 cases of DCPA

Age in Years	Total No.
16 – 20	13
21 – 25	12
26 – 30	16
31 – 35	7
36 – 40	1
41 – 45	1
46 – 50	
51 – 55	

Table II- Gives distribution of 25 cases of Sycosis Barbae

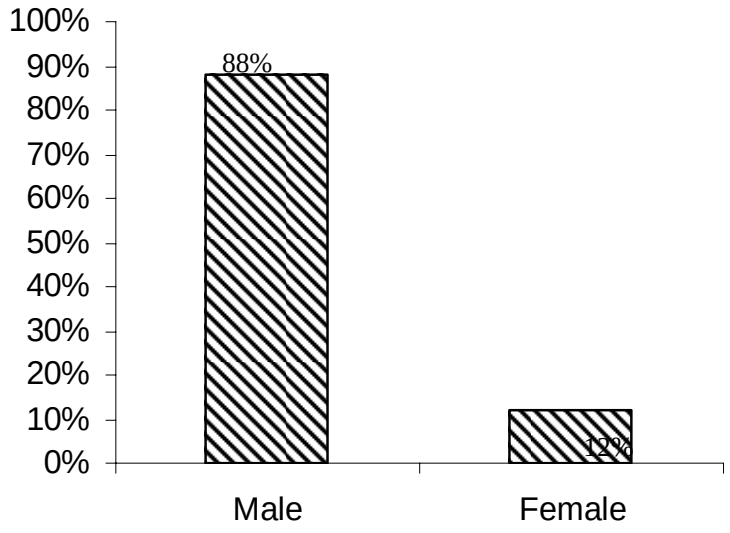
Age in Years	Total No.
16 – 20	
21 – 25	
26 – 30	4
31 – 35	8
36 – 40	5
41 – 45	5
46 – 50	2
51 – 55	1

Most of the cases in the study were between the age of 26 years to 35 years.

82% of cases of DCPA were under the age of thirty. Equal percentage (50%) of patients of Sycosis Barbae was below and above the age of 35 years.

Sex Distribution

75 cases were recorded in total out of which 6 cases of DCPA were female. The incidence of female was 12% in DCPA
Following bar diagram shows sex distribution.



Occupation and Socioeconomic Status

80% of the cases belonged to low socioeconomic strata.

Table III - Shows occupation distribution of all 75 cases

Occupation	DCPA	Sycosis Barbae
Mechanic	9	1
Office	4	6
Mason	8	1
Coolie	7	3
Student	6	1
Hotel Worker	1	5
Farmer	2	1
Tailor	2	2
Printer	2	-
Housemaid	2	-
Textile Workers	1	-
Barber	1	1
Police	2	-
Rickshaw Driver	1	-
Hawker	1	1
Gardner	1	-
Business	-	2
Sweeper	-	1

Males with DCPA were mostly automobile workers (mechanics), while females (50%) with DCPA were office going. In contrast patients with Sycosis Barbae were more of office going (24%).

Precipitating Factors

External oil application was the main precipitating factor in 17 cases (34%) of DCPA Psychological stress was found to be more in patent with Sycosis Barbae.

Seasonal

Significant proportion of cases (80%) showed summer exacerbation.

Duration of the Disease

In 70% of the cases the duration of disease varied from 6 months to 3 years. Shortest duration was 1 month. Longest duration was 5 years.

Table IV - Gives the duration of all 75 cases

Duration in Years	DCPA	Sycosis Barbae
0 – 0.6	12	4
0.7 – 1	17	11
1.1 – 2	10	7
2.1 – 3	6	2
3.1 – 4	4	1
4.1 – 5	1	-
Total	50	25

Symptoms related to cutaneous lesions

Itching with burning sensation was the predominant symptom in DCPA.

While in patients with Sycosis Barbae burning sensation was the prominent complaint followed by inability to shave.

In females, DCPA was more of a cosmetic concern.

Table V- Shows Symptoms in 75 patients (DCPA and Sycosis barbae)

Symptom	DCPA	%	Sycosis Barbae	%
Itching	33	66	12	48
Burning Sensation	29	58	22	88
Pain	10	20	5	20
Cosmetic Complaints	4	8	6	12
Inability to shave*	-	-	13	52

* Inability to shave for patient in Sycosis Barbae

Dermatological Lesions

Lesion in the study varied according to the stage of the disease of the patient. More follicular pustules were seen in acute stage, atrophy and scarring were seen later stages.

Table VI - Shows dermatological lesion of total 75 cases of Sycosis Barbae and DCPA

Lesion	DCPA	%	Sycosis Barbae	%
Pustules	38	76	21	84
Erythema	11	22	8	32
Papules	32	64	12	48
Crusting	16	32	10	40
Pigmentation	12	24	2	8
Scaling	33	66	11	44
Eczema	1	2	4	16
Atrophy	27	54	3	12
Scarring*	3	6	5	20
Alopecia	27	54	15	60
Icthyosis	9	18	3	12

* scaring was especially looked in for in Sycosis Barbae.

Table shows follicular pustule was the predominant dermatological lesion in DCPA and Sycosis Barbae.

Site of Distribution

In 50 cases of DCPA, 80% were confined to lower limb. Six cases showed concurrent involvement of upper and lower limbs. Four patients had both DCPA and Sycosis Barbae at the same time. A single case showed involvement of trunk.

In 25 cases of Sycosis Barbae. only one person had involvement of upper lip only. In all other cases beard region was involved. Below chin (submental and submandibular) area of the beard was the commonest region seen to be affected.

Associated Dermatological lesions

Sycosis Barbae and DCPA was found simultaneously in four patients.

Dermatological lesions associated with DCPA

Psoriasis	-	2 cases
Tinea cruris	-	3 cases
Vitiligo	-	1 case
Milaria rubra	-	7 cases

Dermatological lesions associated lesion with Sycosis Barbae

Seborrheic dermatitis	-	7 cases
Tinea Versicolor	-	4 cases
Milaria Rubra	-	5 cases
Tinea cruris	-	3 cases
Acne	-	1 case
Albinism	-	1 case

Systemic associations

In DCPA one case was found to have diabetes mellitus and four cases lymphadenopathy was seen. One case of Sycosis Barbae was found to be VDRL reactive in 1 : 4 dilution and one case was seropositive for HIV.

Investigation

Complete Haemogram (TC, DC, Hb, ESR)

In 75 cases recorded total count was normal in almost all cases except two cases of Sycosis Barbae showed leucocytosis.

ESR – was found to be normal in all cases.

Blood Sugar – One case of DCPA found to have diabetes mellitus which was later confirmed by glucose tolerance test.

Blood for ELISA – One case of Sycosis Barbae was found to have HIV seropositivity on ELISA test.

Blood for VDRL – VDRL was found to be reactive in 1 : 4 dilution in one patient of Sycosis Barbae. Patient did not manifest any clinical sign or symptoms of syphilis.

Pus Culture and Sensitivity

Pus culture and sensitivity was done in 59 (38 cases of DCPA, 21 cases of Sycosis Barbae) out of 75 cases.

Table VII – Shows the organism cultured in both DCPA and Sycosis Barbae

Organism	DCPA	%	Sycosis Barbae	%
Staphylococcus aureus	37	97.3	17	80.9
Pseudomonas	1	2.6	1	4.7
Proteus	1	2.6	-	-
α hemolytic streptococci	-	-	1	4.7
No Growth	1	2.6	4	19

Nasal swab culture from anterior nares

The test was done to detect carrier state in all cases for staphylococcus aureus.

Eighteen cases of DCPA (36%) and eight cases of Sycosis Barbae (32%). Patients grew Staphylococcus aureus on nasal swab culture.

Pus for Gram staining - Gram positive cocci was seen in 80% cases of DCPA and Sycosis Barbae arranged in grape like cluster.

Tzanck Smear – It was done in only one case of Sycosis Barbae to rule out herpetic sycosis. It was negative

Scraping of fungus – It was done in 5 cases of Sycosis Barbae to rule out dermatophytic sycosis. It was found to be negative in all.

Sensitivity pattern to various class of antibiotic

Sensitivity was tested against various class of antibiotic. The antibiotic against which sensitivity was tested was penicillin, amikacin, ciprofloxacin, ofloxacin, gentamycin, cefotaxime and cloxacillin.

Table VII: The Sensitivity pattern of 54 cases where S.aureus growth was positive is shown.

No.	Class of Antibiotic	Sensitive	%	Resistance	%
1.	Penicillin	4		53	94.4
2.	Ciprofloxacin	45	79.6	12	
3.	Ofloxacin	49	87	8	
4.	Cloxacillin	56	94.1	1	
5.	Gentamycin	47	81.4	10	
6.	Amikacin	50	87	7	
7.	Cefotaxime	48	83.3	9	

DISCUSSION

Age distribution :

This study had patients with an average age between 20 – 30 years for DCPA and 25 – 35 years for Sycosis Barbae. Harman et al[38] observation for DCPA was 18 – 30 age group Sugathan P et al noted patients between 16 – 30 years. In this study age of patients with DCPA ranged from 16 – 30 years which correlates with the literature.

Tony Burns et al[28] noted most cases of Sycosis Barbae in third to fourth decade. In this study most cases belonged to the same age group.

Sex Distributions

Over all study had 6 females. All which had DCPA with incidence of 12%.

Desai et al [46] and Clarke et al [38] noted male predominance in DCPA. Sugathan P et al noted DCPA in females with terminal hair over the shin. In this study 90% of cases of DCPA is male similar to literature. Sycosis Barbae is disease exclusive to male and so was shown in the study[28].

Seasonal incidence

Tropical environment favour DCPA and Sycosis Barbae. More than 50% of cases showed summer exacerbation which is similar to as noted by Sugathan P et al[41,28].

Occupational

Most cases of (18%) DCPA were automobile worker who had direct or indirect history of external oil application as was noted by Sugathan P et al[41].

Sycosis Barbae showed predominance in office going people (24%) which was according to literature, where Sycosis Barbae has been stated to be more indoor worker[28].

Socioeconomic status – Most of the cases (34%) were from the low economic strata which has not been documented before.

Precipitating factors – One third cases of DCPA had external oil application as their precipitating factor which correlated with Sugathan et al studies[41].

Most cases of Sycosis Barbae were found to be associated with underlying psychological stress. Whether this stress had a

indirect role precipitating Sycosis Barbae was not known. No earlier such report has been documented in literature.

Duration of Disease

Both DCPA and Sycosis Barbae are folliculitis with chronic exacerbation and remission. Most of the cases in this study ranged from 6 month to three years. A case with 5 years chronicity was also noted. A case of Sycosis Barbae with 20 years chronicity has been recorded in the literature[36].

Symptoms

Itching and burning sensation was found to be predominant in both DCPA and Sycosis Barbae. It is similar to finding of Sugathan P et al[41] and Meinhof et al[35]. Difficulty to shave was the next common complaint patient had in Sycosis Barbae.

Dermatological lesion

Follicular pustule was the predominant lesion in both DCPA and Sycosis Barbae. Papular lesion with alopecia and atrophy was also noted in (70%) significant cases. Atrophy was seen in more 60% of patient with DCPA. Scarring was notable in some cases of (20%) Sycosis Barbae (lupoid sycosis) which is similar to

literature[28]. Scarring has also documented in DCPA in Sugathan P et al which was supported by this study[41].

Eczema was found in the vicinity of DCPA lesion in 25% of patients. Eczema due to disease per se or irritation due to topical medicament was contemplated.

Distribution

Eighty percent of cases of DCPA were located to lower limb. Some cases had concurrent involvement of forearm and beard which has been documented by Clarke et al[38] and Harman et al[42].

Sycosis Barbae tends to affect the whole beard area. In this study the below chin region of the beard (submental and submandibular) area was most commonly found to be affected. This finding has not been reported before. There was a single case in which only moustache (upper lip) involvement was noted.

Associated Lesions

In four cases of DCPA patient had concurrent Sycosis Barbae milaria rubra was the commonest to associated lesion in DCPA. Seborrheic dermatitis was found to be present in 30% cases of Sycosis Barbae. Greasy and seborrheic skin is noted in Sycosis

Barbae in the literature[28].One case of albinism with Sycosis Barbae has also been noted in the study.

Investigations :

Bacteriology – S.aureus was found to be the major etiological agent in ninety percent cases of DCPA and Sycosis Barbae. These reports are consists with findings of Desai et al[46] for DCPA and Valentine et al[33] for Sycosis Barbae. Pseudomonas, proteus and α hemolytic streptococci were also cultured. Whether they were primary etiological agent or due to secondary infection was not known.

Nasal Swab Culture

Coagulase negative Staphylococci was the most common isolate found from nasal swab culture. In some cases both streptococci and staphylococcus aureus were cultured. 18 cases of DCPA (35%) and 8 cases of Sycosis Barbae (30%) showed S.aureus from culture of nasal vestibule. This correlates well with literature [2,5].

Blood Sugar

One case of DCPA (2%) was found to be diabetic with subsequent glucose tolerance test confirmation. DCPA with diabetes has not been reported.

ELISA – one case of Sycosis Barbae was found to be sero-postive for HIV – 1 antibody. Occurrence of Sycosis Barbae in HIV with increased frequency has not been documented. Increased frequency of seborrheic dermatitis with folliculitis as skin marker finds mention in patient with HIV and needs further study.

VDRL – VDRL study was done in all suspected cases. One patient of Sycosis Barbae was VDRL reactive in 1 : 4 dilution, patient had no previous history of primary or secondary syphilitic lesion nor history of previous treatment Stoke et al[(56)] has reported hypertrophic nodular lesion of face and follicular syphilide on back and extensor in secondary syphilis. But there are no reports of Sycosis Barbae per se. Here VDRL reactivity may be a technical error or patient had coexisting latent syphilis which needs further confirmation by specialized test (FTA – Abs) which was not done in out case.

Sensitivity pattern to various class of antibiotics[57]

Staphyloccocus aureus showed a notable resistance to penicillin in the pus culture and sensitivity. Cloxacillin was found to be invariable most effective antibiotic to which S.aureus was sensitive. It was followed by amikacin, ciprofloxacin and cefotaxime. These findings correlates with the literature. Penicillin G – resistant Staphylococcus aureus strains from clinical infections always produce penicillinase. They now constitute about 90% of Staphylococcus aureus isolate in community in USA[58].

CONCLUSION

Following conclusions were drawn from the study.

1. More common in males
2. Mean age of occurrence between 20 – 40 years
3. Tropical environment exacerbated the disease.
4. DCPA was predominantly found in automobile workers in males and office going females. People with Sycosis Barbae were more of office going.
5. Itching and burning sensation were the predominant complaints.
6. External oil application and psychological stress were the predominant precipitating factors in DCPA and Sycosis Barbae respectively.
7. The disease showed chronic exacerbation and remission over a period of months and years.
8. Lower limbs were more commonly affected in DCPA and submental and submandibular area of the beard area the most common site in Sycosis Barbae.

9. Staphylococcus aureus was the most common isolate from both pus culture and nasal swab culture.

10. Cloxacillin was the most sensitive antibiotics against Staphylococcus aureus.

11. In general patient had no systemic association.

BIBLIOGRAPHY

1. Rooks textbook of dermatology – 7th Ed. / Edited by Tony Burns… et al. Volume 2, Chapter 27, Bacterial infection. Authors R.J. Hay & B.M. Adrians, Blackwell Science Ltd., 2004. Introduction and normal flora of skin, Pg. 27.1 – 27.5.

2. Maple PAC, Hamilton, Miller, Brunfitt W. Worldwide antibiotic resistance in metnicillin – resistant staphylococcus aureus. Lancet 1989; 537 – 40.

3. Hurley H J. Fundamental cutaneous microbiology. Authors Thomas L. Ray, Richard R, Marples. Dermatology 3rd Ed. Philadelphia; W B Saunders; 1992, Pg. 701 – 9.

4. Millian S J, Baldwin J N, Rheins M S et al. Studies on the incidence of coagulase positive staphylococcus in a normal unconfined population. Am J Public Health 1960; 50: 791 – 8.

5. Noble W C. Microbiology of human skin, 2nd Edition, London; Lloyd – Luke, 1981.

6. Wheat J, Kohler R B, White A. Treatment of nasal carriers of coagulase positive staphylococci Jn; Maibach H I, Aly R, eds. Skin microbiology: Relevance to clinical infections, New York; Springer, 1981; 50 – 8.

7. Klempner M S, Styrt B. Prevention of recurrent staphylococcal skin infection with low-dose oral clindamycin therapy. JAMA 1988; 260: 2682 – 5.

8. Hutchison J G P, Green C A, Grimson T A. Nasal carriage of staphylococcus aureus in nurses. J Clin Pathology 1957; 10: 92 – 5.

9. Aly R, Shinefield H I, Strauss W G et al. Bacterial adherence to nasal mucosa cells. Infect Immun 1977; 17: 546 – 9.

10. Fitzpatricks dermatology in general medicine – 6th Edition / edited by Irwin M. Freedberg (et al) vol.2 Chapter 194. Pyodermas. Author Peter K. Lee, Mathew T. Zipolli, Arnold N. Weinberg, Morton Swartz and Richard A. Johnson Pg 1856.

11. Ginsburg C M. Staphylococcal toxin syndromes. Paediatr Infect Dis 1991; 10: 319 – 21.

12. Chen W-Y Sager S, Jung E et al. Human peripheral blood lymphocyte activation by protein A from staphylococcus aureus. Infect Immun 1982, 36: 59 – 65.

13. Neuber K, Konig W. Effects of staphylococcus aureus cell wall products and enterotoxin B on immunoglobulin synthesis and CD^{23} expression in patients with atopic dermatitis. Immunology 1995, 75: 23 – 8.

14. Dahl M V. Staphylococcus aureus and atopic dermatitis. Arch Dematol 1983, 119: 840 – 6.

15. Rooks textbook of dermatology – 7th Ed. / Tony Burns… et al., Volume 2, Chapter 2, Bacterial infection. Authors R J Hay and B M Adrians. Blackwell Science Ltd., 2004. Gram positive bacteria, Pg. 27.6 – 27.8.

16. Jawetz, Melnick and Adelbergs Medical Microbiology 22nd Edn, Chapter 14. The staphylococcus. Appelton and lange 2001 Pg. 197 – 8.

17. Bibel D J, Aly R, Shinefield H R et al. Importance of the keratinized epithelial cell in bacterial adherence. J Invest Dermatol 1982, 79: 250 – 3.

18. Aly R. Bacteriology of atopic dermatitis. Acta Derm Venereol (Stockh) 1980, 92: 16 – 8.

19. Spink M, Strong S. A possible hereditary factor in the nasal carriage of staphylococci. Lancet 1966, i; 1337 – 9.

20. Kinsman O S, McKenna R, Noble W C. Association between histocompatibility antigens (HLA) and nasal carriage of staphylococcus aureus. J Med Microbial 1983; 16: 215 – 20.

21. Burr M L. Staphylococcus aureus and atopic dermatitis – Arch Dermatol 1983, 119: 250 – 3.

22. Verhoef J, Verbrugh H A. Host determinants in staphylococcal disease. Annu Rev Med 1981, 32: 107 – 22.

23. Editorial. Detecting host response to staphylococcal infection. Lancet 1986, i; 953 – 4.

24. Noble W C. Microbial skin disease: its epidemiology. London: Arnold, 1983.

25. Rebora A, Nunzi E, Pezzuolo M et al. Buckley's syndrome. Br J Dermatol 1978, 99: 569 – 72.

26. Rooks textbook of dermatology – 7th Ed. / Tony Burns… et al., Volume 2, Chapter 2, Bacterial infection. Authors R J Hay and B M Adrians. Blackwell Science Ltd. 2004. Inflammatory disorder of hair follicles, Pg. 27.20 – 27.23.

27. Fitzpatricks dermatology in general medicine – 6th Edition / edited by Irwin M. Freedberg (et al) vol.2 Chapter 194. Pyodermas. Author Peter K. Lee, Mathew T.Zipolli, Arnold N. Weinberg, Morton Swartz and Richard A. Johnson Pg 1861.

28. Rooks textbook of dermatology – 7th Ed. / Tony Burns… et al., Volume 2, Chapter 2, Bacterial infection. Authors R J Hay and B M Adrians. Blackwell Science Ltd. 2004. Inflammatory disorder of hair follicles, Pg. 27.24 – 27.25.

29. Dermatology edited by Jean L Bolognia, Joseph L Jorizzo, Ronald P Rapini. Vol. 1, Chapter 40. Folliculitis and follicular occlusion tetrad. Author Paul Kelley, Elsevier Limited 2003. Deep folliculitis, Pg. 460 – 1.

30. J.L. Alibert, Description des maladies de la peau 2nd Edn, Paris, Wahlen, 1825, First Description of Sycosis Barbae in Vol. II, Pg. 214.

31. I ADVL. Textbook and Atlas of dermatology – 2nd Ed. / Editied by R G Valia. Vol. 1, Chapter 11. Bacterial infection. Authors Gurmohan Singh, Vineet Kaur, Sanjay Singh. Bhalani Publishing House, Mumbai, 2001. Pg. 195.

32. Fitz Patrick's dermatology in general medicine – 6th Ed. / Edited by Irwin M. Freedberg… et al. Vol. 2, Chapter 194. Phyodermas: Authors Peter K Lee, Mathew T. Zipoli, Arnold N. Weinberg, Morton N. Swartz and Richard A. Johnson. Pg. 1860.

33. Valentine FCO, Hall Smith S P. Superficial staphylococcal infection. Lancet 1953, 11: 351 – 4.

34. Strauss J S, Kligman A M. Pseudofolliculitis of the beard. Arch Dermatol 1981, 117: 20 – 22.

35. Meinhof W, Braun – Falco. Folliculitis sycosiforms atrophicans. Barbae Hoffmann (sycosis lupoides Milton – Brocq, ulerythema sycosiform unna). Dermatol Wochenschr 1966, 152: 153 – 67.

36. Pinkus H, Rudher E. Sycosis vulgaris agminata (Lutz). Dermatol Wochenschr 1965, 151: 628 – 32.

37. Rooks textbook of dermatology – 7th Ed. / Edited by Tony Burns et al. Vol. 2, Chapter 31, Mycology. Authors R J

Hay and M K Moore. Blackwell Science Ltd., 2004. Superficial and cutaneous mycoses. Pg. 31.30 – 31.31.

38. Clarke, GHV. A note on dermatitis cruris pustulosa et atropicans. Transaction of the Royal Society for tropical medicine and hygiene, 1952; 46(5): 558 – 59.

39. Arnozan X. Folliculitis depilantes despartes glabres. Bull society for dermatology and syphilology, 1987. 3: 187.

40. Miller R F. Epilating folliculitis of glabrous skin. Archives on dermatology, 1956; 83: 177 – 84.

41. Sugathan P et al. Folliculitis eruris pustulosa et atropicans. Indian Journal of Dermatology and Venereology 1973; 39 – 40.

42. Harman R R R M. Dermatitis cruris pustulosa et atropicans. The Nigerian skin disease. British Journal of Dermatology, 1968; 80: 97 – 107.

43. Gary Jules, Brauner. Dermatitis cruris pustulosa et atropicans or segmental folliculitis. Current dermatology therapy, Edited by Stuart Maddin Alastair and Jerone, 1982, Pg. 118 – 199. W B Saunder Company, Philadelphia, London, Toronto, Mexico.

44. Rooks textbook of dermatology – 7th Ed. / Edited by Tony Burns… et al. Vol. 2, Chapter 27, Bacterial Infection. Authors R J Hay and B M Adrians. Blackwell Science Ltd. 2004. Inflammatory disorder of hair follid, Pg. 27.21.

45. Gougerto H. Dermatologica 1950, 7th Edition, Paris, Malione.

46. Desai S C et al. Therapy of resistance pyogenic folliculitis on legs in adult males with hypergammaglobulinaemia. Indian Journal of Dermatology and Venereology 1964; 30: 89 – 97.

47. Wood D C F and Ray Battley F. Effects of various detergents on human epidermis. British Journal of Dermatology 1971, 84: 320 – 23.

48. Pardo Castello V. Dermatology in tropical rural zone. Archives of Dermatology 1956, 74: 115.

49. Lever W F and Gundula S L. Histopathology of skin 1986, V Edition. J P Lippincott Company, Philadelphia, London, Mexico, Pg. 292.

50. Hirschmann J V. Tropical antibiotics in dermatology arch dermatol 1998, 124: 1691 – 700.

51. Wheat L J, Kohler R B, Luft F C et al. Long term studies on the effect of rifampicin on nasal carriage of coagulase – positive staphylococci. RCV infect dis 1983, 5: S 459 – 62.

52. Klemper M S, Styrt B. Prevention of recurrent staphylococcal skin infection with low dose oral clindamycin therapy. JAMA 188, 260: 2682 – 5.

53. Maibach H I, Strauss W G, Shinfield. British Journal of Dermatology 1968, 81: 69.

54. Harrisons principal of internal medicine 15th Edition / Editors Eugene Braunwald et al., Vol.2 Ch.139, Staphylococcal infection. Authors Jeffrey Parsonnet, Robert L. The McGraw Hill companies Ltd. 2001. Antimicrobial therapy for infection caused by Staphylococcus aureus. Table 139 – 2. Pg.898.

55. Kalpana Shenoy, Sriniwas et al. Efficacy of cotrimaxazole and PUVA for management of chronic folliculitis of legs. Indian Journal of Dermatology Venereology And Leprology 1990, 56: 223 – 25.

56. Modern clinical syphilogy edited by John M Stokes. Author H Beerman, N R Ingraham J H, Philadelphia, Oct 1994, Chapter XIII. Clinical aspects of relapse, reinfection and progression in early syphilis Pg. 527 and Pg. 634.

57. Goodman and Gilmans the pharmacological basis of the therapeutic. 10th Edition / Edited by Joel G Hardman, Alfred Goodman Gilman. Chapter 44, 45, 46. Authors William A Petri Jr., Henry F, Chamber. The McGraw Hill Companies, 2001.

58. Jawetz, Melnick and Adelbergs Medical Microbiology 22nd Edn, Chapter 14. The staphylococcus. Appelton and lange 2001 Pg. 202.

59. Klebsiella oxitoca folliculitis mimicking tinea barbae in a diabetic man. Authors Antonio Cascio, MD, Claudo

Guarneri MD. International Journal of Dermatology 2005,44,588-89

MASTER CHART

S. No.	Entity	Age	Sex	Occupation	Season	PPT Factors	CLINICAL FEATURES				Associated Lesion	INVESTIGATION			
							Duration (M/Y)	Symptoms	Sites	Lesions		P & Cs	Culture of Ant Nasal Swab	Sensitivity to Antibiotics	ELISA, VDRL, Scraping , Biopsy & Others
1.	DCPA	28	M	Office	S	-	2Y	I/P	RL, LL	Pa/Pu/S/Pi/Ey	Ecz	SA	SA	S – cef, Amk, Clox, gen, cip, ofl, R- Pe	
2.	DCPA	30	M	Mechanic	S	Oil E/A	3Y	I/B	RL, LL,LT	Pa / AT/A/S/Sc	-	-	NG		
3.	DCPA	32	M	Mason	-	-	6M	I	RL, LL	Pu/Pa/At/S/Ic	MR	SA	NG	S – 2,3,4,5,7 R – 1,6	
4.	DCPA	20	F	Office	-	-	7M	I/C	RL ,LL	Pu/Pa/A/At/C/S /Cd	-	SA	NG	S – 2,3,4,5,6 R – 1,7	
5.	DCPA	24	M	Mason	-	-	1Y	I/B	RL,LL,RT	Pa/A/C/S	Ecz	-	NG		
6.	DCPA	23	M	Farmer	S	-	2Y	I/B	RL,LL,LT, RT	Pa/Pu/A/At/S/C	-	SA	SA	S – 2,3,4 R – 1,5,6,7	
7.	DCPA	18	M	Student	S	-	1Y	B	RL, LL	Pu/S/At/Ic	-	SA	SA	S – 1,2,3, 4, 5, 6, 7	
8.	DCPA	20	M	Coolie	-	Oil E/A	8M	I/P	Rl, LL	Pa/At/S/Ic	Ecz	-	NG		
9.	DCPA	23	M	Coolie	S	-	6M	B	RL, LL	Pa/Pu/C/Ey	-	SA	NG	S – 3,4,5,6 R – 1,2,7	
10.	DCPA	24	M	Mechanic	S	Oil E/A	6M	B	Rl	Pu/Pa/At/S	-	SA	SA	S -2,3,4,5,6,7 R – 1	
11.	DCPA	18	M	Tailor	-	Oil E/A	3M	I/B	RL,LL, LT	Pu/C/A/Ic	Pso	SA / Pr	SA	S – 2,3,4,5,6 R – 1, 7	Diabetic
12.	DCPA	24	F	Mason	S	-	2Y	I/B/P	RL, LL	Pa / Pi / A/ At	-	-	NG		
13.	DCPA	33	M	Mechanic	-	Oil E/A	4Y	B	RL, RF, LF, Bd	Pa/Pi/S/C/At	Ecz	-	NG		
14.	DCPA	40	M	Mechanic	S	-	3Y	B/P	RL, LL	Pa/Pu/At/A/S	-	SA	NG	S – 1,2,3,4, 5,6,7	
15.	DCPA	27	M	Printer	-	Oil E/A	2Y	I/B/P	RL, LL	Pa/Pu/At/A/S	-	SA	NG	S – 2,3,4,7 R – 1,5,6	
16.	DCPA	30	M	Mason	S	-	1Y	I	RL, LL	Pa/Pu/S/Ic	-	SA	SA	S-2,3,4,6 R-1,5,7	
17.	DCPA	24	M	Mason	S	Oil E/A	1M	B	RL, LL, RT, LT	Pa/Pu/S/Ic	MR	SA	NG	S-2,3,4,5,6,7 R-1	
18.	DCPA	30	F	Housemaid	-	Oil E/A	3Y	I/B	RL, LL	Pa/Pu/S/C/Ey	Ecz	SA	SA	S-2,3,4,6 R-1,5,7	

S. No.	Entity	Age	Sex	Occupation	Season	PPT Factors	CLINICAL FEATURES				Associated Lesion	INVESTIGATION			
							Duration (M/Y)	Symptoms	Sites	Lesions		P & Cs	Culture of Ant Nasal Swab	Sensitivity to Antibiotics	ELISA, VDRL, Scraping , Biopsy & Others
19.	DCPA	32	M	Mechanic	S	Oil E/A	3Y	I/B	RL, LL, RF, Bd	Pa/Pi/S/A/At	Vit, MR	-	NG		
20.	DCPA	30	M	Mechanic	-	-	4Y	B/P/C	RL, LL	Pa/A/S/At/Sc	-	-	SA		
21.	DCPA	17	M	Printer	-	-	3M	I	RL, LL	Pa/Pu/S/A/Ey	MR	SA	NG	S-3,4,5,6,7 R-1,2	
22.	DCPA	22	M	Textile	S	-	1Y	I/B	RL, LL, RT	Pa/Pu/S/Ic	-	SA	NG	S-4,5,6,7 R-1,2,3	
23.	DCPA	20	M	Student	-	-	1Y	I/B	RL,LL	Pa/Pu/S/Ey	TC	SA	SA	S-2,3,4,5,6,7 R-1	
24.	DCPA	16	M	Student	-	Oil E/A	3M	B	RL, LL	Pu/A/At	TC, MR	SA	SA	S-2,3,4,5,6,7 R-1	
25.	DCPA	30	F	Housemaid	-	-	1Y	I/C	RL, LL, Abd	Pa/Pi/A/At/Cd	MR	-	NG		
26.	DCPA	24	M	Student	S	-	3Y	I	RL, LL	Pi/C/At/S/Ic	-	-	NG		
27.	DCPA	26	M	Barber	-	-	4Y	I/B	RL, LL,RF	Pu/Pi/At/S/Ey	TC	SA	NG	S-2,3,4,6,7 R-1,5	
28.	DCPA	28	M	Watchman	S	Oil E/A	2Y	B/P	RL, LL, RT, LT	Pu/At/A/S/C		SA/ Ps	SA	S-2,3,4,5,6,7 R-1	
29.	DCPA	30	M	Rickshaw	-	-	1Y	I	RL, LL,LF	Pu/Pa/A/C	-	SA	SA/ Ps	S-2,3,4,5,6,7 R-1	
30.	DCPA	32	M	Police	-	-	3Y	I	RL, LL	Pa/Pi/At/A/S	-	-	NG		
31.	DCPA	18	M	Coolie	S	-	8M	I	RL, LL	Pa/Pu/A/EY	Ecz	SA	NG	S-2,4,5,6 R-1,3,7	
32.	DCPA	19	M	Coolie	S	-	9M	B	RL, LL	Pu/S/C/Ic	-	SA	NG	S-2,3,4,5,6,7 R-1	
33.	DCPA	45	M	Farmer	-	-	2Y	I/B	RL, LL, Bd, M	Pu/Pi/S/C	-	SA	SA	S-2,3,4,7 R-1,7	
34.	DCPA	33	F	Office	-	-	6M	I/C	RL, LL	Pu/S/C/Ey/Cd	-	SA	SA	S-2,3,4,7 R-1,5,6	
35.	DCPA	17	M	Student	-	-	1Y	B	RL, LL	Pu/S/At/C	-	SA	NG	S-2,3,4,5,6,7 R-1	
36.	DCPA	25	M	Mason	-	-	2Y	I	RL, LL	Pu/Pa/A/S	Pso	SA	NG	S-2,3,4,5,6,7 R-1	
37.	DCPA	25	M	Coolie	S	Oil E/A	1Y	I/P	RL, LL, RT	Pa/Pu/A/S	-	SA	NG	S-3,4,5,6,7 R-1,2	
38	DCPA	30	M	Hawker	S	-	4Y	B	RL, RT	Pa/Pi/At/A	-	-	NG		

S. No.	Entity	Age	Sex	Occupation	Season	PPT Factors	CLINICAL FEATURES				Associated Lesion	INVESTIGATION			
							Duration (M/Y)	Symptoms	Sites	Lesions		P & Cs	Culture of Ant Nasal Swab	Sensitivity to Antibiotics	ELISA, VDRL, Scraping, Biopsy & Others
39.	DCPA	22	F	Office	-	-	5M	B/P/C	RL, LL, LT	Pu/S/C/Sc/ Cd	-	SA	SA	S-2,3,4,5,6 R-1,7	
40.	DCPA	18	M	Coolie	S	-	6M	I	RL, LL	Pu/S/C/Ic	-	SA	Sa	S-2,3,6,7 R-1,4,5	
41.	DCPA	19	M	Mason	-	-	2Y	B	RL, LL, RT	Pa/Pu/At/A	-	SA	NG	S-2,3,4,5,6,7 R-1	
42.	DCPA	27	M	Mechanic	S	Oil E/A	5Y	I/B	RL, LL, RT	Pi/Pa/At/A	MR	-	NG		
43.	DCPA	26	M	Hotelworker	-	Oil E/A	3Y	I/B	RL, LL	Pa/Pu/A/At	-	SA	Sa	S-4,5,6,7 R-1,2,3	
44.	DCPA	21	M	Coolie	S	Oil E/A	1Y	B/P	RL, LL, RT	Pu/At/A/S	-	SA	NG	S-2,3,4,5,6,7 R-1	
45.	DCPA	32	M	Mechanic	S	Oil E/A	2Y	I/B	RL, LL, RT	Pa/Pu/S/A	-	SA	NG	S-1,2,3,4, 5,6,7	
46.	DCPA	30	M	Gardener	S	-	2Y	I/B	RL, LL	Pa/Pu/A/At	MR	SA	NG	S-2,3,4,5,6,7 R-1	
47.	DCPA	26	M	Mechanic	-	-	10M	B/P	RL, LL	Pu/At/S/C/	-	SA	NG	S-2,3,4,5,6, R-1	
48.	DCPA	22	M	Mason	S	-	1Y	I	RL, LL, RT	Pu/Pa/Pi/A/ S	-	NG	NG		
49.	DCPA	17	M	Student	-	-	4M	I/B	RL, LL	Pu/S/Pi/Ey	-	SA	NG	S-3,4,5,6,7 R-1	
50.	DCPA	32	M	Police	S	-	1Y	B/P	RL, LL	Pu/S/A/At	-	SA	NG	S-2,3,4,5,6,7 R-1	
51.	Sycosis Barb	32	M	Hotelworker	S	Shaving	1Y	SD	M,Bd	Pu/Ey/S/A	Seb derm	SA	SA	S-4,5,6,7 R-1,2,3	
52.	Sycosis Barb	32	M	Coolie	S	Shaving /stress	4Y	SD/B	M,Bd	Pa/Pu/C/A/ Ic	-	SA	NG	S-3,4,5,6,7 R-1,2	VDRL R 1:4 dil
53.	Sycosis Barb	35	M	Hotelworker	S	Shaving /stress	5M	P/E/SD	Bd. M	Pu/C/S/A/J c	Seb Derm	SA	Ng	S-2,3,4,5,6,7 R-1	
54.	Sycosis Barb	42	M	Office	S	Shaving	2Y	I/B	Bd	Pu/C/A/S/ Cd	TV	NG	SA		Scraping –ve
55.	Sycosis Barb	40	M	Hawker	S	-	2Y	I/B	Bd	Pu/C/Ey	-	NG	NG		
56.	Sycosis Barb	45	M	Tailor	-	-	1Y	B/SD	Bd, M	Pa/Pu/Ey/Ic	TC	SA	SA	S-2,3,4,5,6,7 R-1	
57.	Sycosis Barb	33	M	Barber	S	Stress	11M	P/B/I	Bd, M	Pu/S/A/C/ Ey	-	SA	NG		

S. No.	Entity	Age	Sex	Occupation	Season	PPT Factors	CLINICAL FEATURES				Associated Lesion	INVESTIGATION			
							Duration (M/Y)	Symptoms	Sites	Lesions		P & Cs	Culture of Ant Nasal Swab	Sensitivity to Antibiotics	ELISA, VDRL, Scraping, Biopsy & Others
58.	Sycosis Barbae	31	M	Office	S	Stress	7M	I/B	Bd	Pa/S/Pu/A/ Cd	MR	SA	SA		
59.	Sycosis Barbae	38	M	Hotelworker	S	-	2Y	B/SD	Bd	Pa/Sc/At/Pi	-	-	NG	S-2,3,4,5,7 R-1,6	
60.	Sycosis Barbae	46	M	Mechanic	-	-	3Y	SD	Bd	Pi/Sc/At	TV/MR Seb derm	-	NG		
61.	Sycosis Barbae	47	M	Business	S	-	1Y	I/B	Bd/M	Pa/Pi/Sc/A/ Cd	Tc	-	SA		
62.	Sycosis Barbae	52	M	Watchman	S	-	2M	B/P/SD	Bd,M	Pu/Ey/C/A	-	SA	NG	S-2,3,4,7 R-1,5,6	
63.	Sycosis Barbae	26	M	Office	S	Shaving	5M	B/P/SD	Bd	Pu/Ey/C/S/ Cd	Sed Derm	SA	NG	S-2,3,4,5,6 R-1	Scraping -ve
64.	Sycosis Barbae	28	M	Office	S	Shaving	7M	I/B	Bd	Pu/Ey/Ec/C	-	SA	NG	S-2,3,4,5,6,7 R-1	
65.	Sycosis Barbae	34	M	Hotelworker	S	Shaving	2Y	I/B	Bd	Pa/Pu/Sc/ Ey	Seb derm	SA	NG	S-2,3,4,5,6,7 R-1,3	
66.	Sycosis Barbae	43	M	Coolie	S	Shaving	1Y	B/BD	Bd	Pu/Pa/A/At	Tc	SA	NG	S-2,3,4,5,6,7 R-1	
67.	Sycosis Barbae	41	M	Farmer	S	Shaving	2Y	I/B/SD	Bd,M	Pu/Pa/A/EC	-	SA	NG	S-4,5,6,7 R-1,2,3	
68.	Sycosis Barbae	27	M	Student	-	Shaving	1Y	I/B	M	Pu/S/Ec/A/ Cd	TV/MR	NG	NG		
69.	Sycosis Barbae	37	M	Business	S	-	6M	B	Bd	Pu/Ey/S/Ec/ Cd	Seb Derm	SA	SA	S-2,3,4,5,6,7 R-1	
70.	Sycosis Barbae	36	M	Coolie	-	-	1Y	I/B	Bd	Pu/Ey/S/C/ Sc/ Dis Nodules	Seb Derm / TV	SA	NG	S-2,3,4,5,6,7 R-1,5	Scraping –ve, HIV +ve

S. No.	Entity	Age	Sex	Occupation	Season	PPT Factors	CLINICAL FEATURES				Associated Lesion	INVESTIGATION			
							Duration (M/Y)	Symptoms	Sites	Lesions		P & Cs	Culture of Ant Nasal Swab	Sensitivity to Antibiotics	ELISA, VDRL, Scraping, Biopsy & Others
71.	Sycosis Barbae	32	M	Mason	S	Shaving	2Y	I/B/SD	Bd	Pa/Pu/A/S	MR	SA /α HS	NG	S-2,3,4,5,6 R-1,6	
72.	Sycosis Barbae	32	M	Office	S	-	2Y	I/B/SD	Bd, M	Pa/Pu/Ey/C	-	SA	SA	S-2,3,4,6,7 R-1,5	Scraping -ve
73.	Sycosis Barbae	31	M	Office	S	-	8M	B/SD	Bd,M	Pu/A/S/C	MR	SA /Ps	SA	S-4,5,6,7 R-1,2,3	Diabetic
74.	Sycosis Barbae	43	M	Hotelworker	-	Shaving	9M	B/P/SD	Bd	Pa/A/S/Pu	Alb	NG	Ng		Scraping –ve
75.	Sycosis Barbae	40	M	Sweeper	-	Shaving	3Y	SD	Bd	Pa/Ey/A/S	-	-	NG		Scraping –ve

KEY TO MASTER CHART

Entity

DCPA - Dermatitis Cruris Pustulosa et atrophicans

Symptoms

I - Itching

B - Burning

P - Pain

SD - Shaving Difficulty

CD - Cosmetic Disability

Sites

RL - Right Leg

RT - Right Thigh

LL - Left Leg

LT - Left Thigh

RF - Right Forearm

LF - Left Forearm

Abd - Abdomen

M - Moustache

Bd - Beard

Lesion

Pa	-	Papule
Pu	-	Pustule
Pi	-	Pigmentation (hypo / hyper)
S	-	Scaling
Sc	-	Scarring
C	-	Crusting
Ey	-	Erythema
Ec	-	Eczema
Ic	-	Icthyosis
A	-	Alopecia
At	-	Atrophy

Pus and Culture Sensitivity and Nasal Swab Culture

SA	-	Staphylococcus aureus
Ps	-	Pseudomonas
Pr	-	Proteus
NG	-	No Growth
α HS	-	Hemolytic Streptococci

Association

ECZ	-	Eczema
Pso	-	Psoriasis
Tc	-	Tinea Cruris

Tv - Tinea Versicolor

Vit - Vitiligo

MR - Milaria Rubra

Seb derm - Seborrheic Dermatitis

Sensitivity pattern to antibiotics

1. Pc - Penicillin

2. Cip - Ciprofloxacin

3. Ofl - Ofloxacin

4. Clox - Cloxacillin

5. Gen - Gentamycin

6. Amk - Amikacin

7. Cef - Cefotaxine

R - Resistance

S - Sensitive

PROFORMA

1. Case No :
2. Name :
3. Age :
4. Sex :
5. Occupation :
6. Address :
7. Entity : DCPA / Sycosis Barbae
8. Presenting Complaints :
    i. Itching
    ii. Burning Sensation
    iii. Pain
    iv. Cosmetic Complaint
    v. Discharge
    vi. Other (Specify)
9. Duration :
10. Mode of Onset :
11. Precipitating factors :
    i. External application of oil
    ii. Shaving
    iii. Stress (Psychological)
12. Seasonal exacerbation : - Summer / Winter
13. Past History :
    i. Diabetes
    ii. HIV Status
    iii. VDRL status
    iv. Others

14. General Examination :

15. Systemic Examination :

16. Dermatological Examination :

a. Site

I. if DCPA :i.Right leg, ii.Right thigh, iii. Right forearm
iv.Left leg, v.Left thigh, vi. Left forearm

vii. Abdomen (trunk) viii. Other (Specify)

II. If Sycosis Barbae : Moustache, Beard

b. Lesions Morphology

Papules	Scaling
Pigmentation	Scarring
Pustules	Icthyosis
Atrophy	Crusting
Alopecia	

17. Associated Conditions :

18. Investigations :

Routine : a) Complete Haemogram – Hb
TC
DC
ESR
Platelets

b) Blood Sugar - RBS

c) Pus culture and sensitivity

d) Gram staining

e) Nasal Swabbing for culture.

Additional

DCPA	Sycosis Barbae
Scraping for fungus	Scraping for fungus
	Tzanck Smear (Herpetic Sycosis)
	ELISA
	VDRL

19. Sensitivity Pattern to different class of Antibiotics :

1. Penicillin - R/S
2. Ciprofloxacin - R/S
3. Ofloxacin - R/S
4. Cloxacillin - R/S
5. Gentamycin - R/S
6. Amikacin - R/S
7. Cefotaxime - R/S

COLOUR PAGES

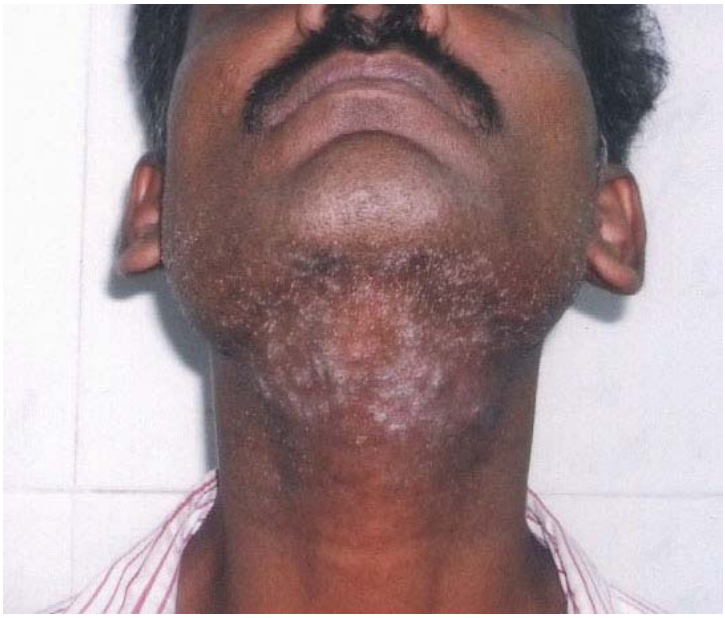


Fig.1. Sycosis Barbae Below The Chin Area (Submental & Submandibular)

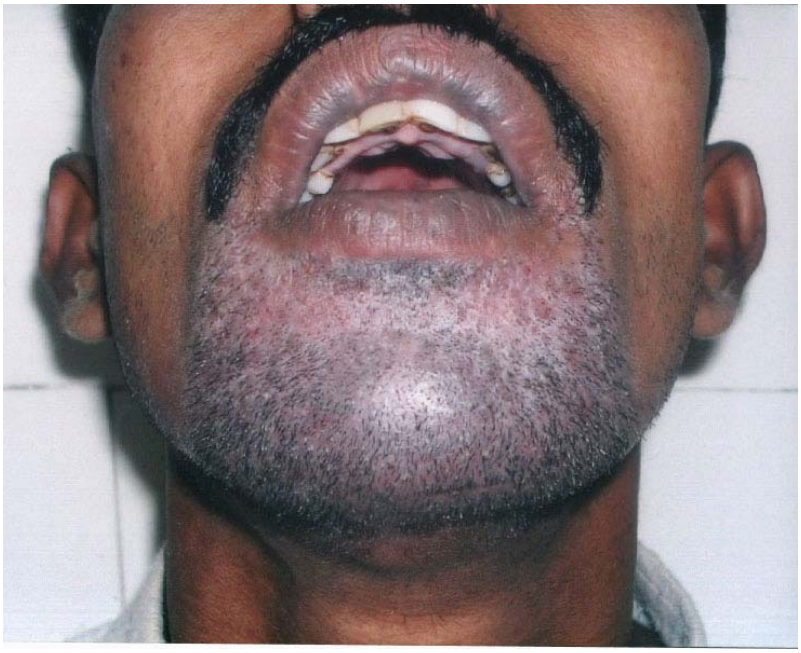


Fig. 2. Sycosis Barbae with Eczematisation

Fig.3. DCPA Over the Lower Limbs

FIG.4. DCPA in a Female Patient

FIG.5. Sycosis Barbae with Scarring (Lupoid Sycosis)

Fig. 6. DCPA
Showing Eczematisation and Scarring

FIG.7. DCPA over the Forearms

FIG.8. DCPA Involving the Lower Limbs and Forearm

FIG.9. DCPA Involving Thigh, Shin and Forearm

FIG.10. DCPA Over The Abdomen

FIG.11. Sycosis Barbae in an Albino Patient

FIG.12. Sycosis Barbae HIV Seropositive Patient

FIG.13. DCPA and Sycosis Barbae in Same Patient

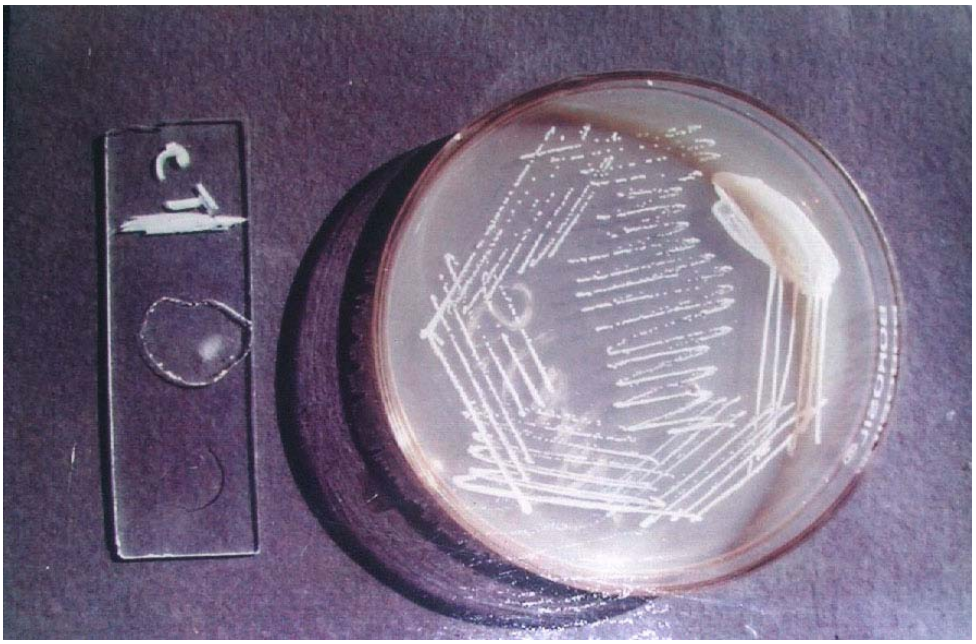


FIG.14. S.aureus Growth on Nutrient Agar.
Slide Shows – Coagulase Positive Test

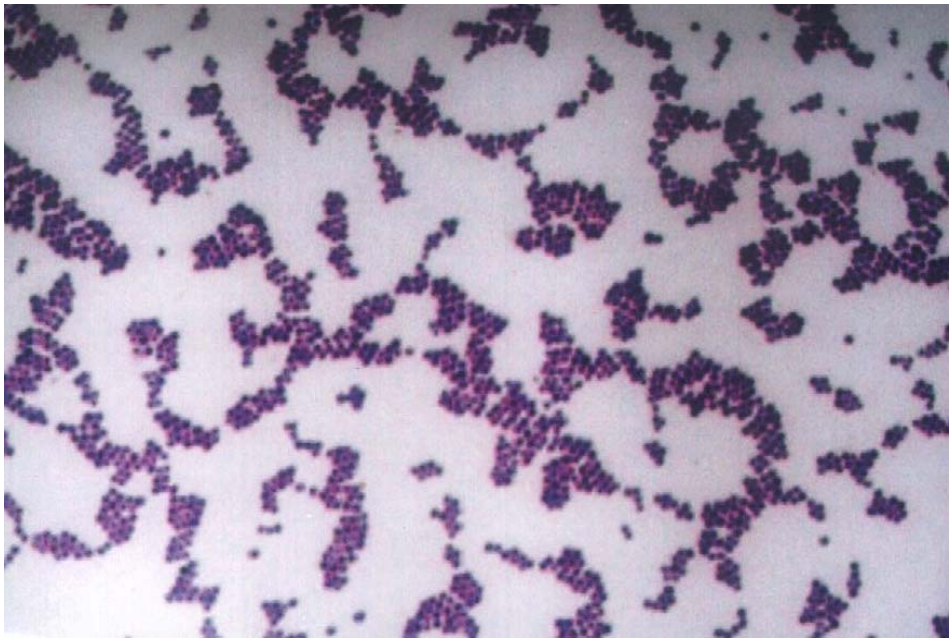


Fig.15. Gram Positive Cocci in Grape Like Clusters in Light Microscopy

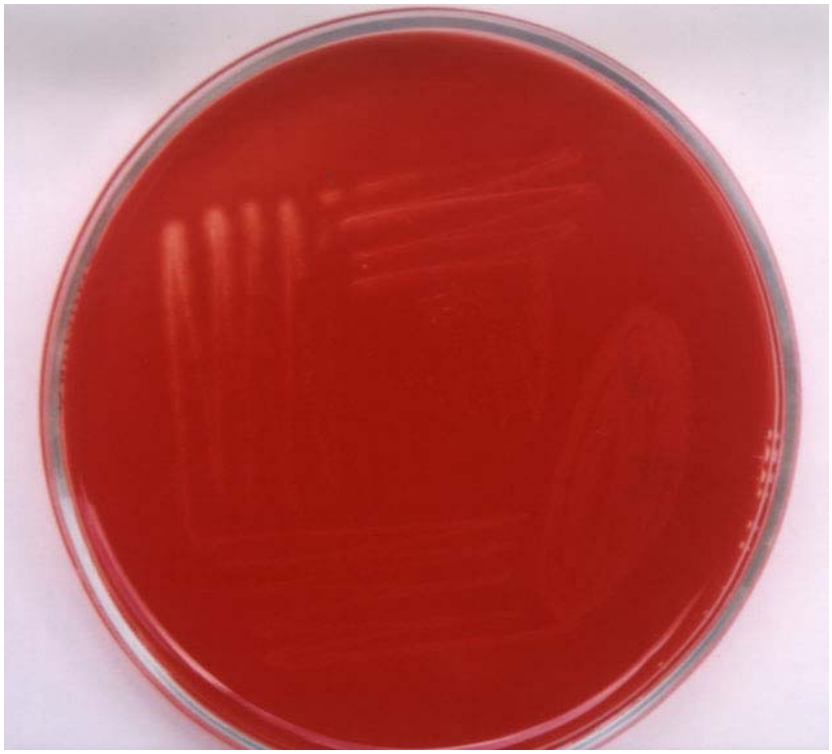


Fig.16. Alpha Hemolytic Streptococci Growth on Blood Agar

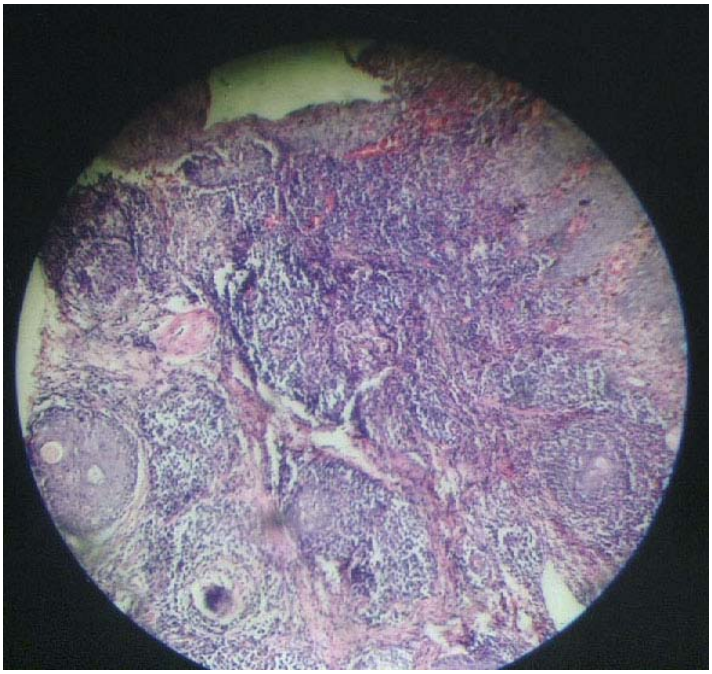


Fig.17. H & E Section Showing Dense Inflammatory infiltrate around hair follicle in Sycosis Barbae

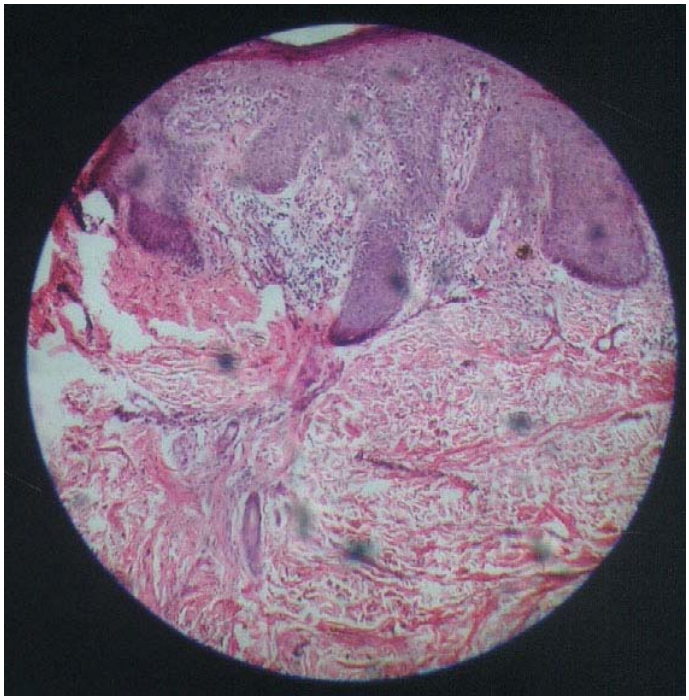


Fig.18. H & E Section Showing Sparse Inflammatory infiltrate around hair follicle in Chronic Stage of Sycosis Barbae

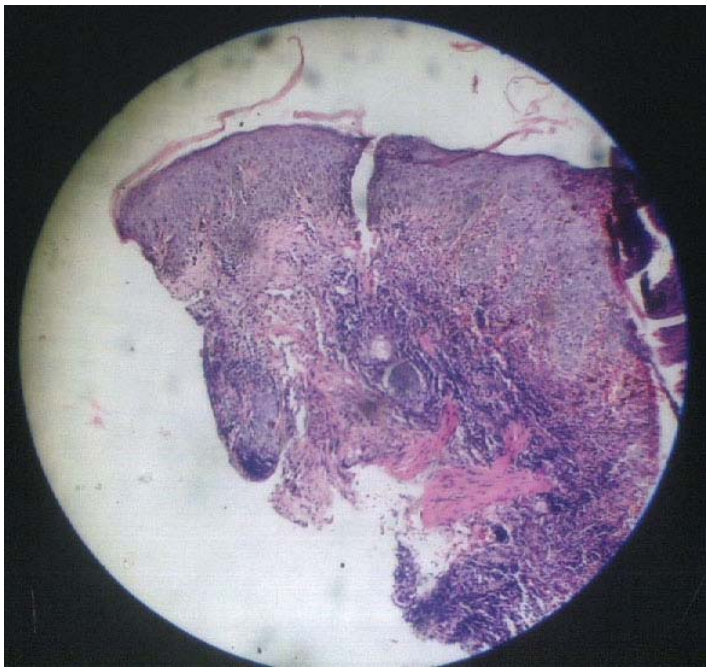


Fig.19. H & E Section Showing Dense Inflammatory infiltrate around hair follicle in DCPA

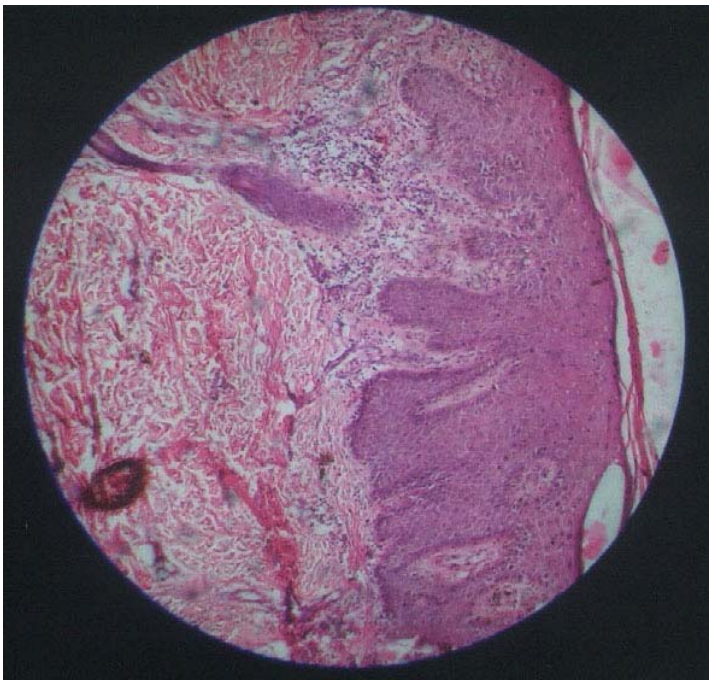


Fig.20. H & E Section Showing Minimal Inflammatory Infiltrate around hair follicle in resolved case of DCPA